kæmia, all due to myelogenous changes, and that these should be known as the myelocytic, lymphoid, and mixed-cell varieties, if we wish to make the blood picture the basis for clinical divisions. Any of these three forms may be either acute or chronic; the myelocytic is usually chronic, the lymphoid usually acute. The nature of the disease, the etiology, and the primary focus in the body we believe to be still undiscovered. Leukæmia as we know it is simply the manifestation of some poison which affects especially the bone marrow, the blood-forming organ in the adult, and ultimately causes death.

REPORT OF A CASE OF SEVERE, PROBABLY PERNICIOUS ANÆMIA, WITH MARKED GENERAL AND CORONARY ATHEROMA, IN WHICH MANY EVIDENCES OF ANEURISM WERE PRESENT WITHOUT ANATOMICAL EXPLANATION FOR THEM.

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Jessie W., a Canadian laborer, aged thirty-one years, married, entered Ward 6 in my Cook County Hospital service March 3, 1897. His previous history was somewhat meagre. Eight years previously he had a single venereal sore two or three months, after which he had rheumatism, and somewhat later an attack of pleurisy, the location of which he could not recall. An alcoholic and tobacco history was elicited.

As to his present history, he had grown weaker, but had not lost any great amount of flesh. Some pain had existed for weeks or months over the lower left chest. The appetite had declined, but anorexia was not absolute, nor did it concern any special variety of food. He vomited once, and eructations of gas were frequent. There had been some slight dyspnœa, increased by exertion and the erect posture. The skin for a week had been somewhat yellow, and the urine was dark, but the bowel movements were not noted. Direct questioning developed no other points.

Physical Examination. The patient lay in a somewhat listless state. His breathing was moderately accelerated, ranging between 25 and 32 respirations a minute. The skin and mucosæ were distinctly yet not deeply icteric. The tongue was covered with a yellowish-white fur. The hair was somewhat thin. Examination in other respects was negative regarding the head, scalp, ears, eyes, and throat. The lungs were wholly negative, except that respiratory excursion was absent over the left lower lobe, laterally and posteriorly. The peripheral arteries pulsated actively, yet arterial tension was low. The initial pulse-rate ran between 90 and 112. The radials and brachials alone were tortuous and rigid to the finger. No inequality in tension nor in the size of the pulse-wave was noticed in any of the arteries, including the femorals.

The heart was normal in its outlines, save that the left border and apex-beat were found in the left nipple line. A soft blowing systolic murmur was heard with equal distinctness over the pulmonic and mitral areas, and the second aortic tone was loud and almost metallic.

Over the left lower chest was observed a diffuse and most distinct pulsation which, as the patient lay on his right side, extended horizontally from the lower angle of the left scapula around the chest into Traube's space and the epigastrium and in a downward direction to a point distinctly below the left costal arch. The pulsation was vigorous and distinctly expansile to both the eye and hand. A systolic bruit was heard over the pulsating area, but was most clearly audible over the left chest behind. No thrill was detected at any time.

The liver's edge was barely palpable on deep inspiration, was even and of normal resistance. The spleen could not be felt. Otherwise, the abdomen was negative. The urine was 1020 c.c.; specific gravity, 1016; a trace of albumin; urea, 1.07 per cent. No sugar, no peptone, etc.; a moderate number of small granular casts was found.

The nervous system was negative.

The stools were not acholic.

The patient was demonstrated in my clinic as aneurism either high in the abdominal aorta or possibly low in the thoracic aorta. Dr. Robert B. Preble, of the attending staff, also examined the case and demonstrated it as aneurism. When in clinic the pulsation could be clearly seen twenty or thirty feet and its striking expansile throb was demonstrated by pasting on the anterior and posterior surface of the lower chest small pieces of bent paper which were widely separated with each cardiac systole. This experiment was performed to demonstrate the expansion to the eye rather than to the hand of the student. The blood was not examined at first but a few days before the patient's death, his temperature, which had occasionally reached 100° and 100.6° F., fell to 97° F. by mouth, his pulse became thready and could scarcely be counted. The respirations became rapid and shallow, with all of the classical symptoms of collapse. Upon reaction the pulsation was less distinct and a decided anæmic appearance was then first noted, possibly having been obscured by the previously icteric hue, which had now largely disappeared. Examination of the femoral and other vessels was carefully made, with the thought that retroperitoneal or other rupture had occurred. The femorals were equal and synchronous with each other and the radials, etc. The blood showed 5000 leucocytes to the cubic millimetre, a differential count of which revealed normal relations. The red cells numbered 1,400,000, and the hæmoglobin by Fleischl's instrument registered 32 per cent. In the stained preparation the red cells varied but little in size and dimension. There was not much poikilocytosis. A large number of nucleated red corpuscles was seen, the great bulk of which were normoblasts, few megaloblasts being detected.

The patient collapsed again and died rather suddenly, March 21st, eighteen days after admission.

The clinical diagnosis was formulated as follows: 1. Arteriosclerosis. 2. Abdominal aneurism, probably pointing toward the diaphragm. Little stress was placed upon the mere fact of the pulsation, but the perfectly clear expansion of the pulsation seemed to establish the diagnosis of aneurism beyond doubt, with rupture explaining the collapse. 3. Dilatation of the left ventricle. 4. Catarrhal icterus. 5. Acute secondary anæmia. The possibility of pernicious anæmia was discussed and considerable uncertainty was experienced, still it seemed to all who examined the case that the evidences of aneurism, followed by collapse from rupture and sudden death, outweighed all other considerations.

Post-mortem. No. 104, March 22d, one day after death, attended by Drs. Preble, Brown, Hammond, Hunter, Champlin, Smith, Humiston, McGrady, Kirby, and the writer.

Some emaciation; well-marked rigor mortis. The pericardium contained 4 grammes of clear serum. The heart weighed 600 grammes; the endocardium presented the tabby-cat appearance; the myocardium was very pale. Very small yellow plaques were found at the base of the mitral valves and in the beginning of the aorta.

In the left coronary artery, one inch below its origin, was a long plaque or series of plaques of yellow color, hard, some elevated and the size of a pin-head, while others were diffuse and larger. Similar changes were found in the right coronary one and one-half inches below its origin. Over the entire length of the abdominal and thoracic aorta were scattered small yellow areas, some of which were hard, rough, elevated, and breaking with a distinct sound and almost nodular.

Nowhere were any bulgings nor aneurism of the aorta. Similar changes existed in the common iliacs, external and internal iliacs, the femoral arteries and a few of the larger branches of the latter. The nose, larynx, and trachea were negative. The bronchial glands were slightly enlarged and on section anthracotic. The mediastinum was negative. The right pleural cavity contained 300 c.c. of clear serum and no adhesions existed. A few old, thin adhesions were found over the left apex, and the base of the left lung was adherent by well-organized tissue to the chest wall and the diaphragm. The adhesions were œdematous. Above these basal adhesions was a small amount of clear serum. The right lung weighed 530 grammes, the left lung 660 grammes. The lungs floated, crepitated throughout, and on section appeared light gray, a frothy yellow serum being readily expressed, particularly from the left lower lobe. The tongue and the pharynx were negative.

The œsophagus and stomach were exceedingly pale, as was the mucosa of the small and large gut, save for some hypostatic post-mortem discoloration. Small, hard plaques similar to those found in the aorta were seen in the superior mesentery artery and its larger branches. The peritoneum was smooth, and the abdominal cavity contained no fluid. The bile-ducts were patent, and the gall-bladder slightly distended with somewhat viscid bile. The liver weighed 1940 grammes, and its surface was light red and mottled. No adhesions were found, and the liver's edge was smooth and sharp. Its consistence was firm, and on section contained but a moderate amount of blood. On section the lobular markings were fairly distinct, the centre showing red and the periphery light. The pancreas was negative, weighing 120 grammes. The spleen weighed 180 grammes, and there were some perisplenitic adhesions, but to the diaphragm only. The organ was dark purple in color, and the parenchyma soft and friable. The left kidney weighed

200 grammes, the right 160 grammes. The capsule was easily stripped, leaving a smooth, very pale, and slightly mottled surface. The section was very pale, the cortical markings were obliterated, and the cortex and medullary portion were as 1 to 3. The pelvis, ureters, bladder, prostate, seminal vesicles, and urethra were negative.

The brain weighed 1650 grammes, and its examination was wholly negative, especial attention having been directed to the bloodvessels. The spine and cord could not be examined.

The long bones could not be examined, but the section of the sternum showed the reddish jelly-like marrow seen in pernicious and other severe anæmias.

The anatomical diagnosis read: Atheroma of the coronary arteries, mitral valves, aorta and its main lower branches; left ventricle hypertrophy and dilatation; fatty degeneration of the myocardium; pulmonary œdema; partial left adhesive pleuritis; chronic nephritis; acute splenic tumor; perisplenitis; marked anæmia of the alimentary tract, etc., pernicious anæmia (?).

The case presents no remarkable pathological features. The vascular changes were very marked for a person aged thirty-one years, and may be referred with some degree of correctness to syphilis. While the blood findings were not exactly characteristic of pernicious anæmia, still they corresponded fairly well with the subacute or milder recurrent type of pernicious anæmia as described by Cabot and others. The exact cause of the collapse, considered by us as due to aneurismal rupture, which led to sudden death in its second occurrence, may have been due to extensive bilateral coronary disease or to this cause combined with the profound anæmia.

The chief clinical interest of the case centres in the pulsation. The pulsation was extensive, attended by a bruit, abdominal as well as thoracic, and markedly expansile.

At first thought it might seem that the pulsation resembled Broadbent's sign, but the pleural adhesion was purely basal, and there was neither pericardial synechia nor indurative mediastinitis. When a pulsable movement, says Broadbent, is seen in the lower posterior chest it may appear at first sight to be expansile, but on more careful scrutiny it is found that there is a tug upon the false ribs during the systole and a sharp rebound during the diastole, etc.

It has been impossible for me to look over the literature bearing upon this unique case, for a lack of a title covering its phenomena. Personal inquiry of a number of experienced clinicians, as Professor Osler, has not developed any similar instance.

The pleural adhesions and lack of respiratory excursion at the base of the left lung was noted clinically, but did not excite any remark, since among the lung changes incident to aneurism, pleurisy with effusion, adhesive pleurisy, lung induration, etc., have been repeatedly described by writers on aneurism since the classical work by

Stokes. The adhesions were thought to be either due (1) to aneurism near the diaphragm, or (2) to mere coincidence. Nothing suggested pulsating neoplasm of the lung or pleura. A pulsating pneumonia as described by Graves was merely mentioned in the clinic as one of the causes of thoracic pulsation, as were pulsations of the liver and spleen in Basedow's disease, aortic and tricuspid regurgitation as rare causes for abdominal pulsation. Mention was then made of the empyeme pulsatile (Aran), the pulsating empyema of Stokes, and the empyema necessitatis pulsans of MacDonnell.

Dr. J. C. Wilson has reviewed with great care the subject of pulsating pleural effusions.

The literature of pleuritis pulsans brings out the fact that most instances (about 92 per cent.) are left-sided. A review of the causes suggested concerns:

1. The theory of Traube, which referred the pulsation to the condition of the heart and pericardium.

2. Comby's theory placed stress upon adhesions to the pericardium and compression of the lung.

3. Féréol's explanation was pneumothorax with a large effusion, these conditions being imperative.

4. Wilson's conditions: (*a*) Some paresis of the intercostal muscles, as results from a long-standing pleuritis; (*b*) local necrosis and perforation of the parietal pleura (empyema necessitatis); (*c*) pneumothorax; (*d*) atelectasis of the lung, which is in relation with and adherent to the pericardium.

Aneurism has often been thought of, but the statement constantly recurs that it can usually be excluded, except when an encysted empyema lies close to the heart. Among the conditions just named practically the only one in our case was the pleural adhesions over the base of the left lung. I have been able to reach no satisfactory explanation of this pulsation, although the active arterial pulsation might have been propagated by means of the adhesions to the chest and by means of the perisplenitic connective tissue to the abdomen. This suspicion, however, in no way accounts for the *expansile character of the pulsation*, which of yore has been held to invariably (ref. 14 and 15) exclude dynamic pulsation.

In the discussion, Dr. Lafleur, of Montreal, recounted a case in which the pulsation was exactly similar to our case, and other signs, as hæmatemesis, paralysis of the left vocal cord, etc., were present. The pulsation was expansile, but the diagnosis of aneurism was incorrect, the autopsy showing gastric ulcer.

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THE PATHOLOGY OF PERNICIOUS ANÆMIA, WITH SPECIAL REFERENCE TO CHANGES OCCURRING IN THE HÆMOLYMPH NODES (EIGHT AUTOPSY CASES).[1]

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The pathology of pernicious anæmia as given in the most commonly used text-books of to-day contains but scant reference to the lymph glands and no mention at all of the hæmolymph nodes. Though much attention has been paid in recent years to the changes occurring in the spleen and bone marrow, the lymphatic structures have been practically disregarded as a possible factor in the pathogenesis of this disease.

In Barclay's case of "Death from Anæmia" (1851) no mention is made of changes in the lymph glands.

The classical description by Addison (1855) states that in "idiopathic anæmia" there is no "renal, splenic, miasmatic, glandular, strumous, or malignant disease."

In one of Wilks' cases (1857) the lymph glands were described as "healthy." Lebert (1858) does not mention the lymph glands. Bristowe, in the same year, reported a case in which the bronchial, mesenteric, and other abdominal glands were described as "healthy." No mention of lymph-gland changes is made by Cazenave in 1860. In the same year Habershon states that there is no lymphatic disease in "idiopathic anæmia." Grohe (1861) does not mention the lymphatic glands

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[1] Read by title at the meeting of the Association of American Physicians, Washington, D. C., April 29, 1902.